Valves

Introduction

The six conditions presented in this lesson are either disorders of valves or things that cause murmurs. The goal is to cover what causes murmurs in general, how physical exam maneuvers alter preload and afterload, and how those changes affect the sound of each murmur. This is the physiology of murmurs in general. By the end of this lesson, you should recognize each of the lesions clinically by sound and by changes on physical exam, know their common etiologies, and be able to interpret their pathophysiology on a Wiggers diagram. We include the Wiggers diagram as an extra. It is left out of the video on purpose (as described in the last lesson).

We start by defining turbulence, then discuss the effects of physical exam maneuvers on preload and afterload and how they affect murmurs. Next, we cover the four main valvular lesions: mitral stenosis, mitral regurgitation, aortic stenosis, and aortic regurgitation, then the two valvular lesions that disobey the rules and are "sound-alike" lesions: HOCM (sounds like aortic stenosis) and mitral valve prolapse (sounds like mitral regurgitation). During the discussion of mitral stenosis, we diverge to cover acute rheumatic fever and its complication, chronic rheumatic heart disease.

Turbulence Makes Noise

Normal blood flow is laminar, occurring linearly through the vessels and across valves. Laminar flow does not make noise. In contrast, **turbulent flow** is nonlaminar and noisy. Hearing turbulent flow with a stethoscope while listening to the chest over a valve is called a **murmur,** and hearing turbulent flow with a stethoscope over a blood vessel is called a **bruit.**

There are two main ways of getting turbulent flow. Turbulence is defined by **flow over surface area**. The greater the flow or the lesser the surface area, the worse the turbulence and the louder the murmur. That means that if you get your heart beating stronger and faster (increased flow) but have a normal valve (normal area), you can provoke a heart murmur. There is no pathology, but the murmur is heard. If, while studying this material (flow normal), you put your stethoscope gently on your femoral artery and take a listen, you won't hear anything. If you press firm and deep into the femoral artery while listening (decreasing area), you can provoke a bruit. **Turbulence generates sound.** Turbulence grows with **increasing flow or decreasing area**. We are going to stay valve-focused for the rest of the lesson and not discuss bruits again (we chose that example because you can do it on yourself). That means that a large defect (large area) may make not a lot of sound, despite being a more severe defect.

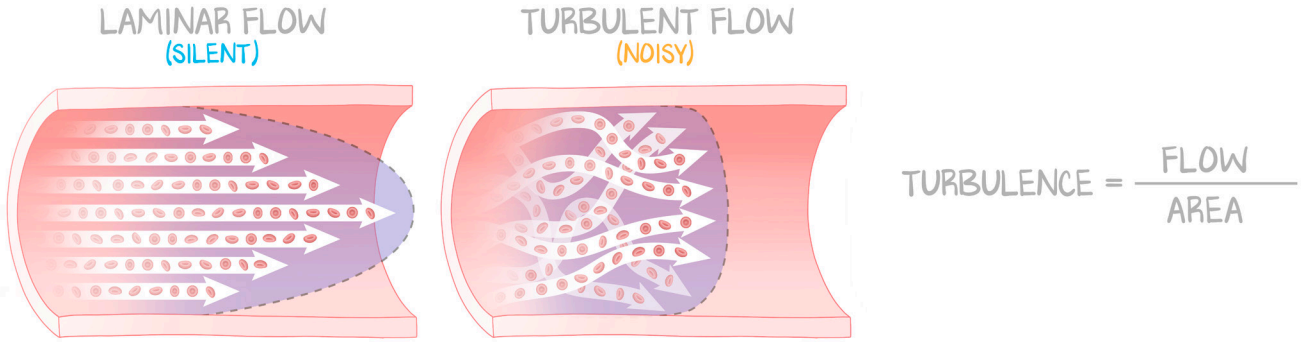

Figure 3.1: Turbulent Flow
(a) Normal laminar flow does not make noise. Turbulent flow does. A smaller area creates more turbulence and more noise. More flow over an area, even if laminar, can generate turbulence and make noise.

Murmurs are graded on a scale of 1 to 6. We don't want to get too far off track (we're touching on clinical reasoning rather than mechanisms of disease), but it helps to have exposure to the clinical tracking tool before you get started. You won't be evaluated on this table until Clinical Sciences.

GRADE	DESCRIPTION
1	Murmur is softer than S_1 and S_2
2	Murmur is as loud as S_1 and S_2
3	Murmur is louder than S_1 and S_2
4	There is an additional thrill
5	Murmur can be heard with stethoscope off chest + thrill
6	Murmur can be heard without Stethoscope + thrill

Table 3.1: Grading Murmurs
Although more of a clinical skill than medical knowledge, we feel obligated to give a proper education in grading murmurs. The failure of clinicians to reliably auscultate has led to the propagation of sloppiness in medical education. "A cardiologist can hear" vs. "a student can hear" is bogus. A cardiologist without a stethoscope, after an AK-47 discharges next to her ear without ear protection, is unlikely to hear a murmur as well as a medical student with normal hearing and a $600 stethoscope. We also recommend that learners invest in a quality stethoscope, even if they are expensive (good ones are over $150, great ones are $300, electronic ones are $600). A farmer can do better work with a tractor than with a plow; a learner can hear a murmur better with a fancy scope than the throwaway version. Knowing the equipment is solid negates any heuristic that the stethoscope is to blame and pushes the learner to evaluate themselves.

Starting Off Right

In Clinical Sciences, you will be expected to hear a murmur, then work back to the diagnosis or etiology. That is also what you will do in practice. When learning it for the first time, here in the Basic Sciences, that doesn't work so well. And if you learn it correctly here, working from the valve lesion to the murmur, it will be easy to go backward when asked to do so later in your career. Start with the valve. Ask what it does during systole and diastole. Whatever the answer, a lesion of that valve will do the opposite of what it is supposed to do.

1. Start with the auscultation point
2. Consider what the valve is supposed to do in systole or diastole
3. Name what it would fail to do in systole or diastole
4. Listen for S_1 and S_2
5. Confirm by identifying the murmur's specific sound and changes with physical exam maneuvers

1 Through 4. There are **four auscultation positions**. In this lesson, we will only be discussing the **aortic** (second intercostal space, right sternal border) and **mitral** (apex of heart, fifth intercostal space, midclavicular line). Whatever you learn for the aortic, apply to the pulmonic; whatever you learn for the mitral, apply to the tricuspid. **Start with the valve** based on where your stethoscope is. Do not start considering murmurs from the cardiac cycle. If you put your stethoscope on the mitral valve auscultation position, you are listening to the mitral valve. There are only two options—stenosis or insufficiency.

Because the mitral valve closes in systole, a murmur during systole must be regurgitation. Because the mitral valve opens in diastole, a murmur during diastole must be stenosis. NOW find S_1 and S_2. Does the murmur occur after S_1 or after S_2? You can get the answer correct without recognizing the sound of the murmur or changes with physical exam. This is insufficient (lol) for practicing medicine (which is why the remainder of this lesson exists), but it is a really safe place to start.

This lesson is going to feel redundant on purpose. It engrains the process—valve position first, thought exercise of what it is supposed to do in systole, what it is supposed to do in diastole, then find S_1 and S_2.

Complex 5. You will need to recognize what the valve lesion sounds like—each has its own unique pattern. In addition to matching the sound, an added complexity is physical exam maneuvers, which are usually designed to change preload or afterload.

Preload (volume, sodium aldosterone, EDV) **is increased by squatting** (compressing the veins of the legs) and **leg lift while laying down** (draining the venous pooling). **Preload is decreased by Valsalva maneuver** (increased intrathoracic pressure temporarily pushes away the venous return). The right heart valves are more affected by changes in preload because they are directly connected to the inferior vena cava and because there is a capillary bed, across which any perfusion pressure from the right ventricle is spent. However, because all of the right-heart cardiac output quickly reaches the left ventricle, maneuvers that affect preload can also be used on the left-sided valves. For almost every murmur (exceptions are mitral valve prolapse and HOCM), more blood in the heart means more flow across the lesion, and therefore a louder murmur. Squatting makes the murmur worse, and standing makes the murmur better.

Afterload (systemic vascular resistance, α_1, AT_1, calcium channels) **is increased by handgrip maneuver**. Only the left-sided valves are sensitive to afterload. Handgrip increases systemic vascular resistance but does not influence the pulmonary circulation. A person cannot significantly alter the vascular resistance of the pulmonary vasculature willingly or with a physical exam maneuver. For this reason, afterload maneuvers will not affect right-sided valves. The significance of this is actually minimal. If you were listening for a right-sided lesion (pulmonic or tricuspid), you would be auscultating in the pulmonic or tricuspid position. You would already know it is right sided, so you wouldn't bother performing a handgrip maneuver.

We specifically call out physical exam maneuvers and their utility in diagnosing the lesions.

Applying the Rules Broadly—Valves Behaving Poorly

Bad valves can be loose and floppy (and so fail to close), or they can be stiff and rigid (and so fail to open).

Bad valves that are too loose and floppy should close, but don't. The result is that blood flows backward through the open valve. The diagnosis for valves that should close but don't is either **insufficiency** or **regurgitation** (these names are synonyms). The chamber that changes is always proximal to the damaged valve. Atria only know how to dilate, so a mitral insufficiency would lead to atrial dilation. Ventricles respond to excess preload with dilation. Both mitral insufficiency and aortic insufficiency will cause the chamber proximal to the lesion to dilate. Insufficiency is usually the result of infection or infarction. There are more details to come.

Bad valves that are too stiff and rigid should open, but don't. This is referred to as **stenosis**. Blood still moves with the flow of the cardiac cycle, but less of it moves. The chamber that changes is always proximal to the damaged valve. Atria only know how to dilate, so mitral stenosis would lead to atrial dilation. Ventricles respond to excess afterload with concentric hypertrophy. Because aortic stenosis impacts the left ventricle uniformly, there will be concentric (all the way around) hypertrophy (sarcomeres added in parallel). Stenosis is the result of rheumatic heart disease or calcification. There are more details to come.

The **mitral valve** (stand-in for the atrioventricular valves) is supposed to **open in diastole** and **close in systole**. A failure to open is called stenosis. A failure to close is insufficiency. The murmur will be heard after the heart sound that defines diastole or systole. Because the mitral valve should close in systole, and a failure to close is insufficiency, mitral regurgitation will be heard after the systolic heart sound, S_1. Because a mitral valve should open in diastole and failing to open is stenosis, mitral stenosis will be heard after the diastolic heart sound, S_2. The mitral valve auscultation point is located at the fifth intercostal space, midclavicular line. If severe enough, mitral lesions (stenosis and regurgitation) can lead to **dilation of the left atrium**. Dilation of the atrium causes stretching, which can result in **atrial fibrillation**. The left atrium is the most posterior heart structure and abuts against the esophagus. Dilation can lead to compression of the esophagus, leading to **dysphagia**. Compression of the recurrent laryngeal nerve can lead to **hoarseness**.

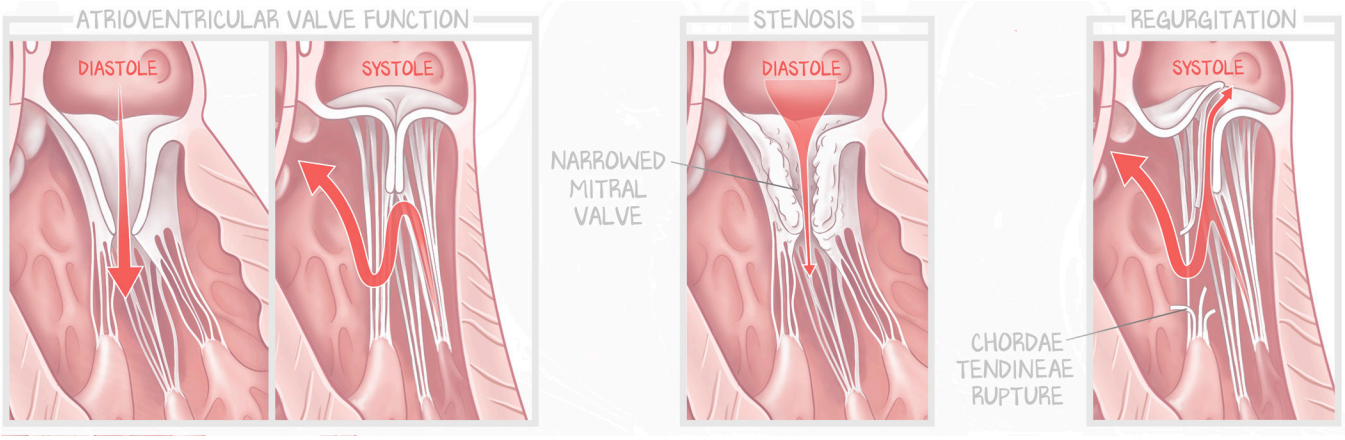

Figure 3.2: Atrioventricular Valve Function and Failure

The **aortic valve** (stand-in for the semilunar valves) is supposed to **open in systole** and **close in diastole**. A failure to open is called stenosis. A failure to close is insufficiency. The murmur will be heard after the heart sound that defines diastole or systole. Because the aortic valve should close in diastole and a failure to close is insufficiency, aortic regurgitation will be heard after the diastolic heart sound, S_2. Because the aortic valve should open in systole and failing to open is stenosis, aortic stenosis will be heard after the systolic heart sound, S_1. The aortic valve auscultation point is located at the second intercostal space, right sternal border.

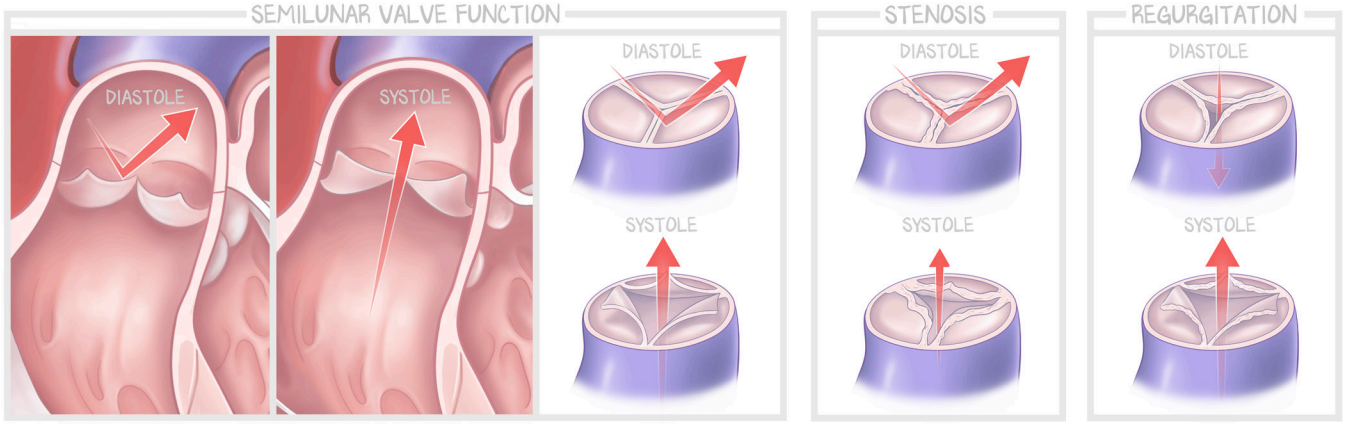

Figure 3.3: Semilunar Valve Function and Failure

Now we go over the lesions one at a time, discussing etiology, murmur sounds, and complications that can arise.

Specific Valve Lesions #1: Mitral Regurgitation

The mitral valve opens during diastole and closes during systole. Because regurgitation is a problem of the valve closing, and the mitral valve closes in systole, mitral regurgitation yields a **systolic murmur**. The murmur is known as a **holosystolic murmur—**one that prevents the auscultation of S_1 and S_2 and is a constant sound throughout. The murmur is best heard at the **cardiac apex** (mitral location) and **radiates to the axilla**.

Mitral regurgitation is caused by infection or infarction. **Infection** of the mitral valve (bacterial endocarditis) can lead to either **perforation** of the valve leaflet or **rupture of the chordae tendineae**. Infection causes the rupture of the tendineae, whereas infarction causes the rupture of a papillary muscle. **Infarction** is a heart attack, because of which the papillary muscle could either fail to contract or rupture altogether if its vascular supply is compromised (muscles have high oxygen demand). Each time the ventricle contracts, blood is ejected into both the aorta and left atrium. The atrium sees more volume than it should, and the atrium responds the only way it knows how—dilation. An echocardiogram will reveal a dilated left atrium and a regurgitant jet through the mitral valve. Treatment is surgical.

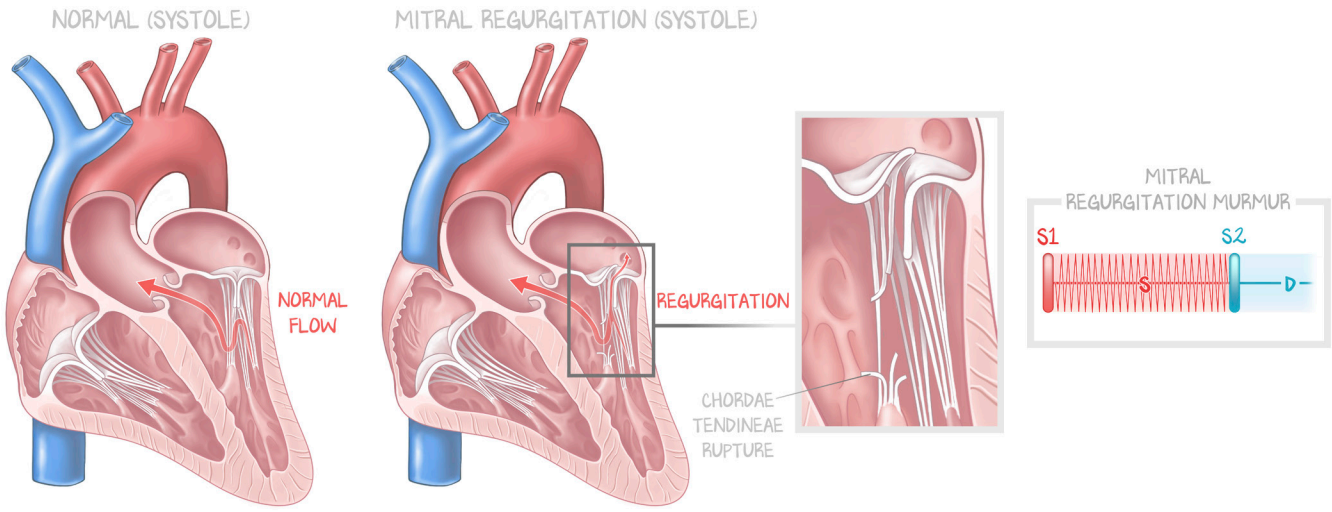

Figure 3.4: Mitral Regurgitation
The ventricular contraction both forces blood up and out the ventricular outflow, through the aorta, and pushes the mitral valve into the left atrium. The papillary muscles prevent the valve leaflets from passing into the atrium. If the chordae tendineae or papillary muscle ruptures, the valve leaflet will be pushed into the atrium, enabling blood to pass into the atrium.

There is another way mitral regurgitation can occur—dilated cardiomyopathy. In mitral valve regurgitation, the left atrium sees a higher volume, so it dilates in response. In dilated cardiomyopathy, all four chambers dilate. As they do, the opening in the fibrocollagenous atrioventricular septum is pulled apart. At some point, the mitral valve leaflets will no longer cover the entire gap—the valves are cardiac tissue and do not proliferate.

Specific Valve Lesions #2: Mitral Stenosis

The mitral valve closes during systole and opens during diastole. Because stenosis is a problem of valve opening, and the mitral valve opens during diastole, mitral stenosis yields a **diastolic murmur**. It is described as a **diastolic rumbling murmur with an opening snap**. The snap is the result of the leaflets

prematurely reaching their maximum open position. Normally, valves do not make a sound when opening. When pathologic, the mitral valve does. The sooner after S_2 the snap occurs, the sooner they have reached their maximum range, the more stenotic the lesion, the worse the lesion. The **earlier the snap, the worse the lesion.** The snap is not caused by the flow of blood over a diminished area, the murmur is. The blood flow through the mitral valve is under low pressure—gravity. Because the pressure is low, the flow is slow. According to the turbulence equation, low flow will make less noise. Because the flow is low for the duration of diastole, the murmur is only a **low rumbling murmur** that doesn't change much throughout diastole. Because there is increased resistance to the atrial kick, the atria respond the only way they know how—**dilation**. An echocardiogram will reveal isolated mitral dilation, a small valve area, and higher pressure than normal. Treatment is surgical—replace the valve.

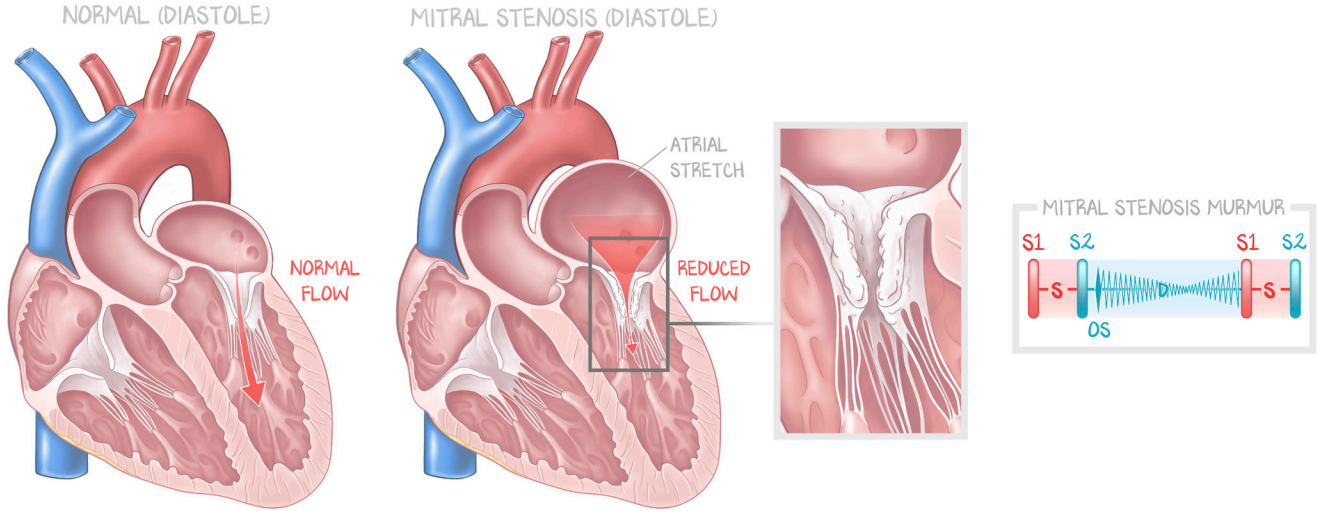

Figure 3.5: Mitral Stenosis
A normal, nonstenotic valve has no difficulty allowing blood into the ventricle. The stenotic valve shows reduced flow because of the tightened opening. It also places backpressure on the atrium, leading to atrial stretch. A closeup of the two leaflets (sectioned anteriorly) shows the rigid and fibrotic valve. Mitral stenosis has an opening snap (OS) then a rumbling diastolic murmur.

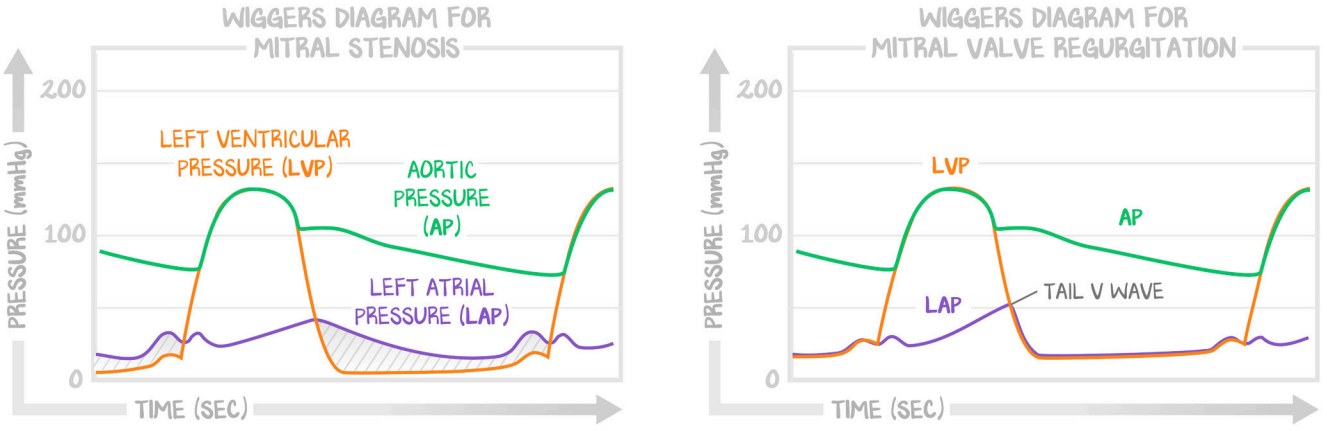

Figure 3.6: Wiggers Diagrams for Mitral Stenosis and Mitral Regurgitation
Mitral stenosis demonstrates elevated left atrial pressure during diastole, indicated by the area under the purple curve, marked in grey. This pressure gradient only exists because the valve area is too small. Mitral regurgitation also causes elevated atrial pressure, but during systole, when the regurgitant jet strikes the left atrium from the ventricle.

Detour to Chronic Rheumatic Heart Disease

Chronic rheumatic heart disease (RHD) is virtually the only cause of mitral stenosis. RHD is a sequela, a progression of **acute rheumatic carditis** (rheumatic fever, RF), an acute, immune-mediated disease that occurs a **few weeks after group A *Strep. pyogenes* pharyngitis** (strep throat). Treatment of strep throat with penicillin has essentially eradicated RHD, and therefore mitral stenosis, in countries where such treatment is standard. RF requires a group A *Strep. pyogenes* pharyngitis infection, lack of penicillin treatment, and genetic predisposition for the disease. Antibodies to the M protein or streptolysin O protein on Group A *Strep*. cross-react with heart tissue (molecular mimicry). The presence of **antistreptolysin-O antibodies** (↑ **ASO**) helps confirm a previous strep infection, a requirement to employ the Jones criteria. Acute RF is diagnosed with the **Jones criteria**, requiring any two major criteria or any one major and two minor criteria.

JONES	MAJOR CRITERIA	MINOR CRITERIA
Joints	Migratory polyarthritis of large joints	Arthralgia
♥	Pancarditis—peri-, myo-, and endocarditis	Fever
Nodules	Subcutaneous nodules	Elevation of serum acute phase reactants
Erythema marginatum	Erythema marginatum	
Sydenham chorea	Sydenham chorea	

Table 3.2: Jones Criteria for Acute Rheumatic Fever

The predominant clinical manifestations of RF are carditis and arthritis. **Arthritis** typically begins with migratory polyarthritis (accompanied by fever), in which one large joint after another becomes painful and swollen for several days before resolving spontaneously. **Carditis** can mean inflammation of the pericardium (pericarditis, pericardial friction rubs), myocardium (myocarditis, elevated troponins), or endocardium (noninfectious endocarditis). Sydenham chorea is a neurological disorder with involuntary, rapid, purposeless movements.

If a biopsy is taken during an episode of acute RHD histology will demonstrate **Aschoff bodies—** fibrinous collagen amidst cardiac myocytes with giant cells and activated macrophages called **Anitschkow cells**. These findings are pathognomonic histologic findings for RF. **Re-exposure to group A *Strep. pyogenes* pharyngitis will provoke reactivation of the immune system**, and similar symptoms can be expected with a recurrent attack. Myocarditis, hallmarked by Aschoff bodies, can lead to fulminant cardiac failure and death (< 1% of cases).

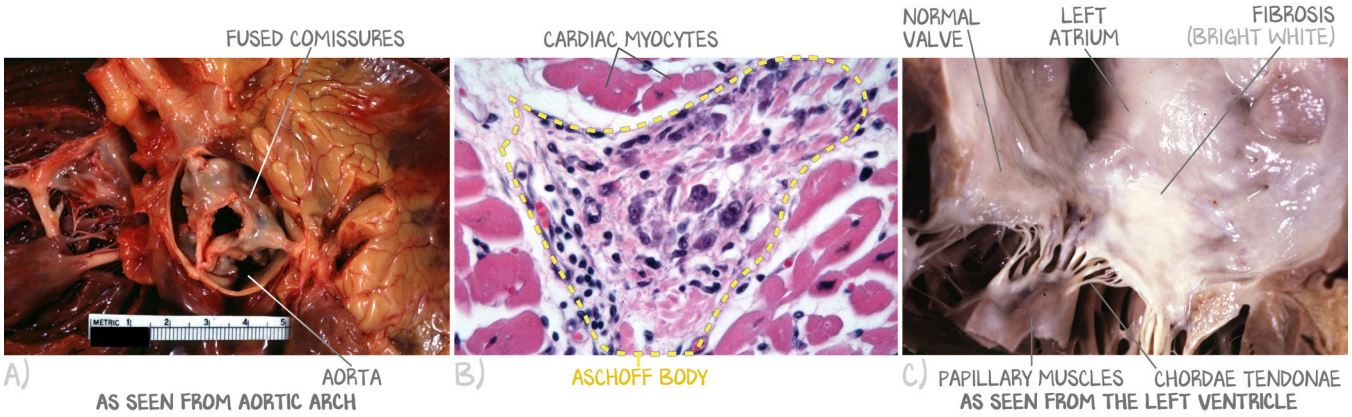

Figure 3.7: Rheumatic Fever
(a) Aortic valve, as seen from the aortic arch, showing the fish-mouth deformity caused by the fusion of the commissures. (b) Aschoff body surrounded by cardiac myocytes and their connective tissue. This is pathognomonic for rheumatic fever (and is usually only seen on autopsy, as a biopsy is not warranted for those who survive). (c) Use the papillary muscles and their chordae tendineae to orient you to the mitral valve above. Notice the endocardium of the atrium is continuous with and the same color as the endocardium of the mitral valve, except where there is evidence of fibrosis. A fibrotic valve will appear white, more opaque than the surrounding endocardium.

Inflammation of the cardiac valve results in progressive stenosis. RHD can affect the mitral or aortic valve. For the mitral valve, **thickening of the leaflets and tendinous cords** results in a stiff, nonmotile valve. For the aortic valve, calcification (from repeated trauma of closure) and fibrous bridging across the valvular commissures create a "fish-mouth" or "buttonhole" appearance, secondary to a **fissure of the commissures**.

Specific Valve Lesions #3: Aortic Stenosis

The aortic valve closes during diastole and opens during systole. Because stenosis is a problem of valve opening, and the aortic valve opens during systole, aortic stenosis yields a **systolic murmur.** It is described as a **crescendo-decrescendo** murmur that **radiates to the carotids**. The valve is stiff and requires more force to open. It is as if there is very high blood pressure in the aorta, as we saw in the lesson on myocardial work. There, increasing the systemic vascular resistance increased the opening pressure of the aortic valve. Here, the increased opening pressure is actually inherent to the valve. In both cases, the valve opens and blood is ejected. When blood is ejected through a stenotic (narrowed) aortic lumen, a murmur is created. But remember how the ventricular ejection curve looked? The pressure continues to increase as the volume decreases. The peak ejection is between the opening and closing of the aortic valve. At first, a little blood passes through—low flow, small murmur—gradually increasing as the ventricle reaches maximum contraction. At maximum contraction, a lot of blood passes through the lesion—high flow, large murmur. From there on, until the valve closes, the flow decreases. This is what gives the murmur its "diamond-shaped appearance," getting louder for the first half of the murmur and then softer for the back half of the murmur. Because there is resistance to blood flow exiting the ventricle, the ventricle responds as it does when there is increased afterload—concentric (uniform around the ventricle) hypertrophy (sarcomeres added in parallel, fatter myocytes). The fatter myocytes cause diastolic dysfunction with impaired filling.

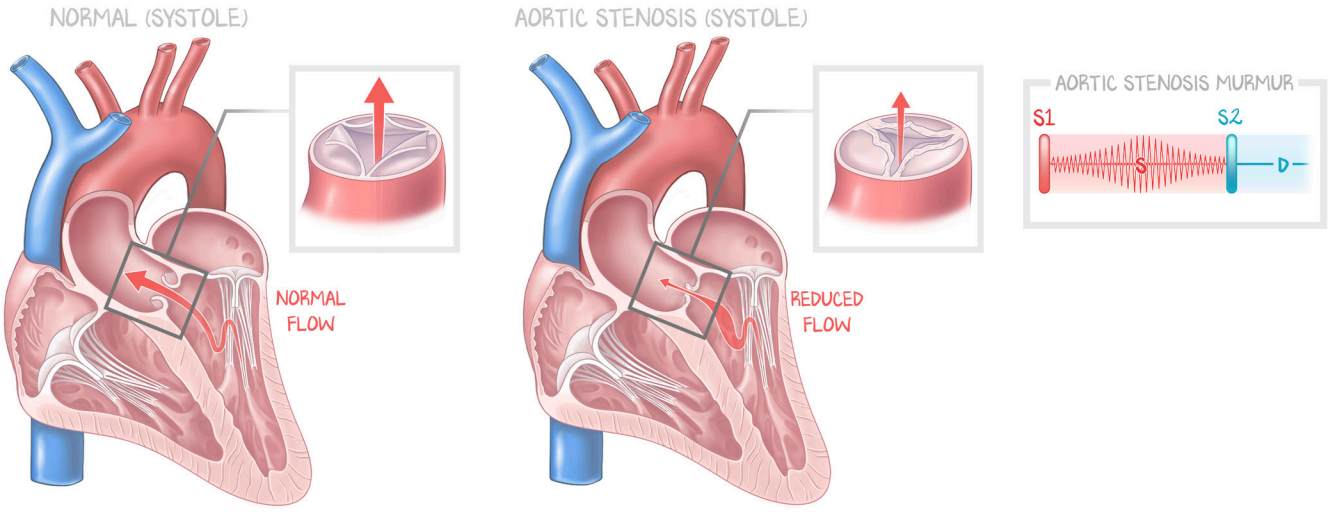

Figure 3.8: Aortic Stenosis
Normally, the aortic valve opens during systole. If it fails to open fully, the same amount of blood is ejected under much higher pressure. The most blood moves across the lesion at maximal contraction, giving the murmur a crescendo-decrescendo quality.

Most of the time, aortic stenosis is the result of wear and tear on the aortic valve. Each time the valve closes, the leaflets slam into one another. The consistent and unrelenting trauma induces the formation of calcifications. Each leaflet remains independent of the others (compared to in RHD), but the leaflets themselves aree stiff and fail to open as they should in systole because the calcifications stiffen the endocardium. The patients most at risk for developing calcifications and aortic stenosis are **old men with coronary artery disease risk factors**. Calcification of the aortic valve has a similar pathogenesis to atherosclerosis, and the ostia of the coronary vessels are behind the aortic valve (more on this in Electricity #1: *Coronary Vessels and Cardiac Conduction*). Simply put, patients with aortic stenosis likely have **exertional symptoms** (chest pain, dyspnea, or presyncope), and a physical exam reveals the murmur. One physical exam finding associated with aortic stenosis is **pulsus parvus et tardus**—a pulse that is weak and late. If you listen to the heartbeat, there is a delay (tardus) between the heartbeat and palpating the pulse at the radial artery, and that pulse is relatively weak (parvus). An echocardiogram will reveal a thickened left ventricular myocardium, a small valve area, a high-velocity jet, and no regurgitation. Surgery for the valve can be traditional open-heart surgery or use newer tools with a transarterial approach, performing the procedure within the lumen of the aorta (TAVR, TAVI, not something you need to know at this stage, just interesting information).

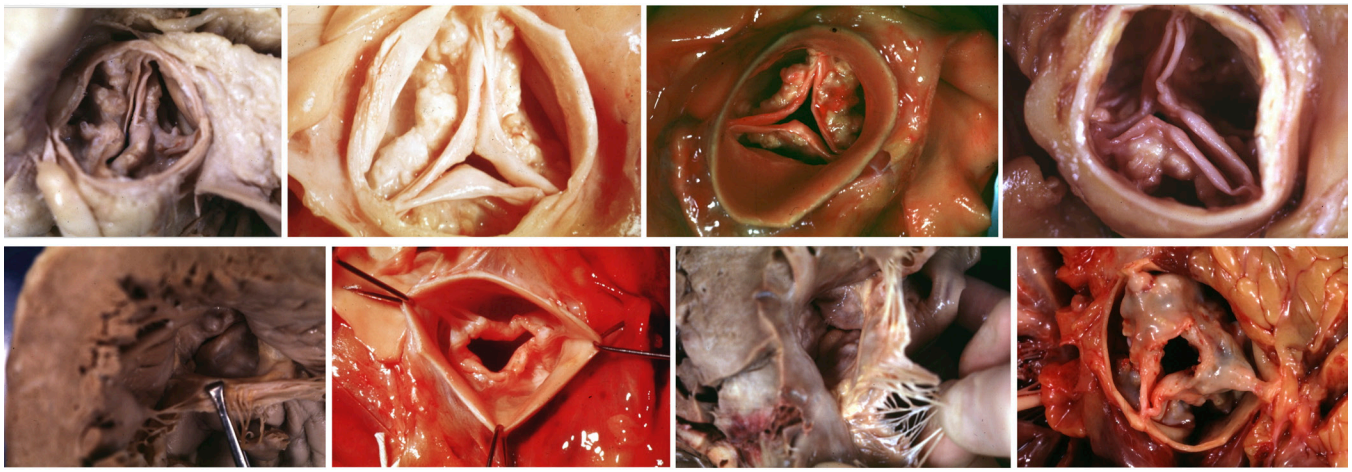

Figure 3.9: Aortic Stenosis in Gross
Multiple views of differently affected aortic valves. Each is from a different autopsy. Aortic valves can be bicuspid (two leaflets), which results in the valve developing calcifications faster than a normal, three-leaflet aortic valve. Most aortic valves have thin leaflet edges that are not fused at the commissures. In rheumatic heart disease, one or all of the commissures may be fused. In some patients, there can be moderately thickened but unfused leaflets (usually of unknown etiology).

Aortic Regurgitation

The aortic valve closes during diastole and opens during systole. Because regurgitation is a problem of valve closing, and the aortic valve closes during diastole, aortic regurgitation yields a **diastolic murmur**. It is described as a **decrescendo murmur**, loudest at the onset of diastole and gradually softening until it's inaudible. Diastole begins when systole ends, when the greatest amount of blood has been ejected into the aorta (stroke volume). During systole, some of that blood provided perfusion pressure, sending the blood down the arterial tree, and some of it distended the aorta. Now in diastole, the aorta recoils, providing perfusion pressure to the blood in its lumen. The aortic valve should close, preventing the backflow of blood into the ventricle and ensuring the aorta's recoil sends blood down the arterial tree. If there is any aortic insufficiency, the defect will not change size—the area of the lesion is fixed. What changes over the duration of diastole is flow. The aorta begins diastole in its most distended state, storing both the most blood and the most force. As aortic recoil continues throughout diastole, there is progressively less blood and, therefore, less force applied to the lumen. The driving force pushing the blood through the defect gets progressively weaker the longer it pushes; thus, the flow across the lesion progressively decreases. Therefore, the murmur is loudest at the onset of diastole (S_2) and softest at the end of diastole (just before S_1).

Like mitral regurgitation, there could be either **infection** or **infarction** leading to failure of one of the valve's leaflets, but as there are no chordae tendineae or papillary muscles, these cannot be the case in aortic insufficiency. Although that is the main mechanism of mitral valve regurgitation, and the aortic valve can suffer the same fate, aortic regurgitation is typically secondary to **aortic root dilation**. Aortic root dilation occurs in retrograde progression of **syphilitic aortitis** (low socioeconomic classes, thoracic aneurysms) and **Marfan aortitis** (tall, lanky white guys with hyperflexible joints and aortic dissection). Anything that pulls the aorta apart can stretch the root, forcing the leaflets to be too far apart to close. Dilated cardiomyopathy, much like mitral regurgitation, can result in aortic insufficiency.

Aortic regurgitation results in **hyperdynamic circulation**. When any ventricle contracts, its stroke volume—the amount of blood ejected with the contraction—is influenced by the end-diastolic volume (a more optimized overlapping of sarcomere filaments). In aortic insufficiency, there is no difficulty in ejecting blood—more blood into the heart, more blood out—but there is difficulty keeping blood out.

During diastole, blood from the aorta falls back into the left ventricle. In other words, some of the blood that the heart just expelled (stroke volume) comes back in through the aortic valve while new blood enters (appropriately) through the mitral valve. More blood into the heart, more blood out—the stroke volume is large because the ventricle is filled through both the aortic and mitral valves. But then, some of the stroke volume falls back into the ventricle. The large stroke volume provides tremendous perfusion pressure, pushing blood down the arterial tree, leading to high systolic pressure. But the aorta loses some of its perfusion pressure that would have driven the blood forward down the arterial tree to the faulty aortic valve. Thus, there is **very high systolic pressure** but then **very low diastolic pressure**. The higher systolic and lower diastolic pressure is called a **widened pulse pressure**.

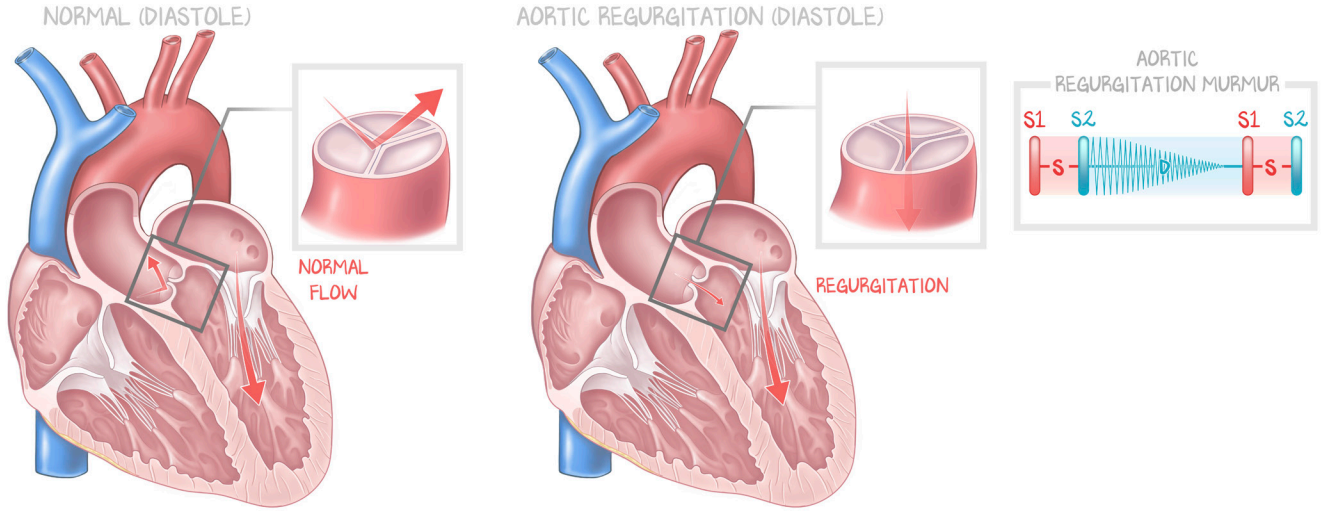

Figure 3.10: Aortic Regurgitation
Normally, the aortic valve closes, preventing the backflow of blood. In aortic regurgitation, there is a gap in the leaflets that allows blood through. Because the greatest flow occurs at the beginning of diastole, the murmur is loudest then, getting progressively softer until it's inaudible.

Widened pulse pressure has some physical exam findings present only in late, severe disease that is generally not seen in the United States (it gets fixed), but that indicate the pathophysiology, and so are often found on licensing exams. One is called a **water-hammer pulse** (brisk upstroke during systole that is rapidly lost during diastole). The **head bobbing** with each heartbeat is the most severe form of a water-hammer pulse, the pressure during systole knocking the patient's head back, then alleviating the pressure over the course of diastole. Like all valvular disease, surgical replacement tends to be the means to correct it.

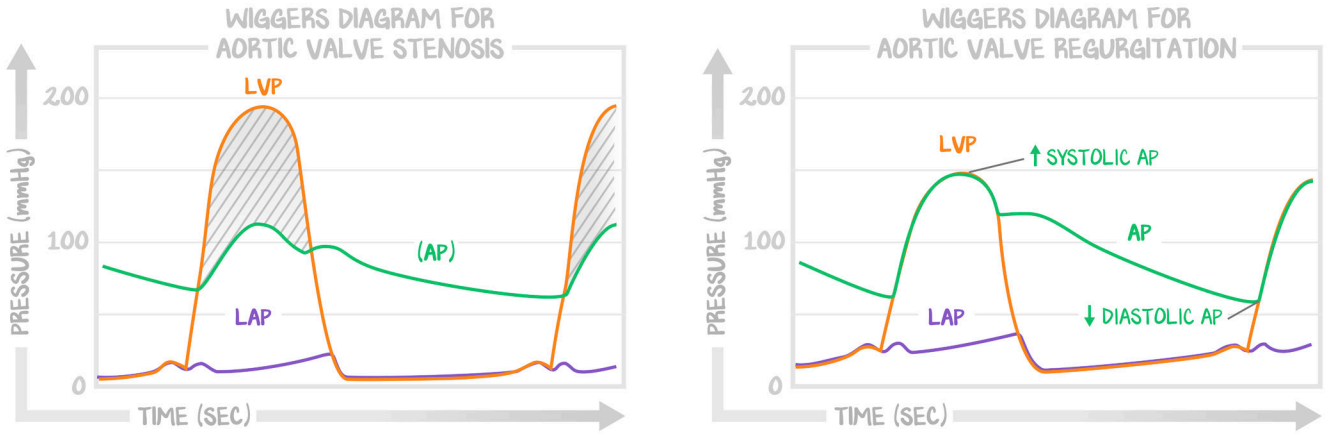

Figure 3.11: Wiggers Diagrams for Aortic Lesions
Aortic stenosis shows an elevated left ventricular pressure, which is much higher than normal and higher than the aortic pressure—the barrier to the ejection of blood is the aortic valve, not the systemic pressure. Therefore, decreasing the aortic pressure (systemic vascular resistance) doesn't help the left ventricle—the afterload it experiences is the valve, not the systemic vascular resistance. In aortic regurgitation, the left ventricular pressure peaks with the aortic pressure (more blood in, more blood out), but then the systemic vascular resistance (aortic pressure, AP) declines gradually over diastole, through the aortic valve.

Mitral Valve Prolapse—MVP

MVP is the tricksy mitral valve regurgitation; it sounds similar to mitral valve regurgitation because it can eventually lead to a regurgitant jet, but it does not obey "more blood, more murmur." In fact, more blood in the heart, less murmur for MVP.

MVP is also known as myxomatous degeneration. That means that the normal fibrous, tough valve is replaced with a soft jelly-like substance. The valve is intact but is flimsy. This lesion's etiology is poorly understood, but it is associated with Ehlers-Danlos and Marfan syndromes, implicating collagen-vascular components. When the left ventricle contracts, the mitral valve remains closed—the edges of the leaflets touch—but wherever the chordae tendineae do not specifically pull it down, the valve balloons back into the atrium. When it reaches its maximum stretch, it clicks. Much like snapping open a parachute, the **systolic click** that defines this lesion is caused by the valve as it snaps open. The systolic click is followed by a "**holosystolic murmur**" that sounds just like mitral regurgitation, only it isn't from S_1 to S_2, but rather from the click to S_2.

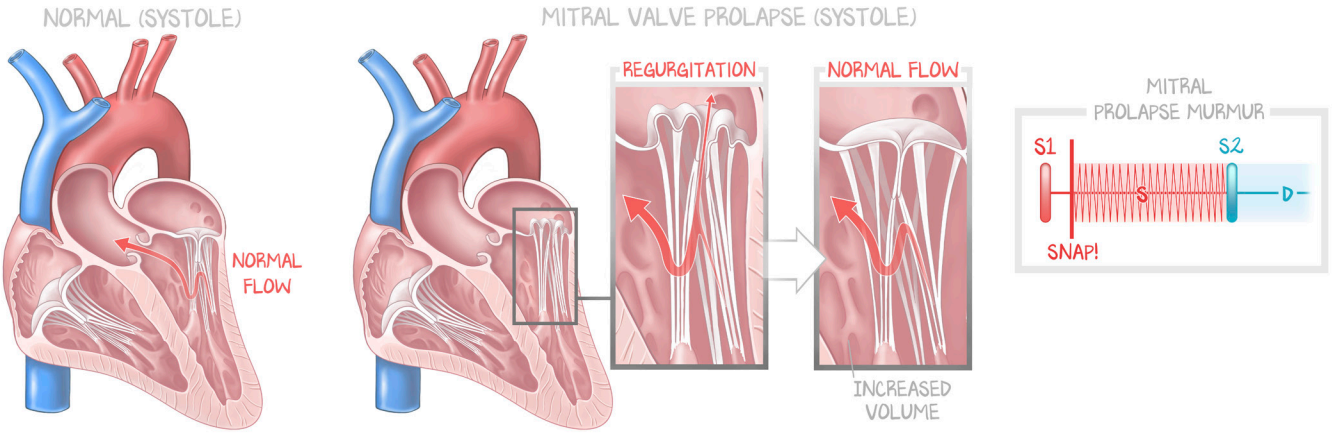

Figure 3.12: Mitral Valve Prolapse
Normally, the contraction of the ventricles pushes blood up and out through the aorta while the papillary muscles resist that force, preventing the mitral valve leaflets from going into the atrium. In mitral valve prolapse, the chordae tendineae and papillary muscles function, but the valve balloons into the atrium, allowing some blood to regurgitate through the valve into the atrium. The murmur sounds like mitral regurgitation, except that it begins after an opening snap.

Hypertrophic Obstructive Cardiomyopathy

Hypertrophic obstructive cardiomyopathy (HOCM) is the tricksy aortic stenosis. In HOCM, **all of the valves are normal**. However, there is effective aortic valve stenosis—subaortic stenosis of the outflow tract from the left ventricle into the aorta. The patient presents with a **crescendo-decrescendo murmur**, but he is a young male athlete (not an old man), AND the murmur gets better with more blood in the heart (as opposed to aortic stenosis, which increases in intensity with more volume). The HOCM patient is almost always male in a vignette, whereas mitral valve prolapse is almost always in a female patient. This is discussed as a diagnosis in greater detail in Structure and Function #8: *Cardiomyopathy*.